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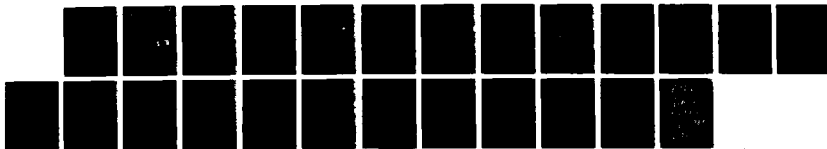
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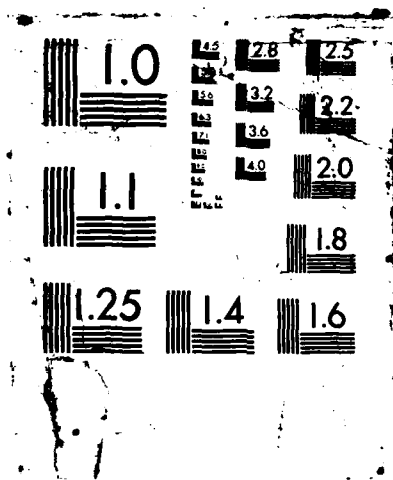
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THE VITE MODEL: A NEURAL COMMAND CIRCUIT FOR GENERATING ARM AND ARTICULATOR TRAJECTORIES

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1. MOVEMENT PLANNING IN MULTI-JOINT SYSTEMS

A major issue in research on the neural basis of motor control is the nature of movement planning in systems with many degrees of freedom; for example, an arm with many controlling muscles acting at several joints, or a speech system with many articulators. All solutions to the planning problem depend upon assumptions about both the mechanics of the effectors and the sensory and computational resources. For example, if an arm has few mechanical degrees of freedom, then the serial preplanning required to work around the arm's inherent constraints becomes a salient issue. Alternatively, if the arm has many degrees of freedom, the computational load imposed by the need for simultaneous coordination becomes a salient issue. If the arm is part of a body that grows, or if a robotic arm must remain in service without external maintenance despite unpredictable changes in its mechanical parameters, then yet another issue comes into view: autonomous recalibration.

This paper extends earlier results (Bullock and Grossberg, 1986, 1988a) that are part of a larger program of research on adaptive sensory-motor control in biological systems (e.g., Grossberg, 1978a, 1982; Grossberg and Kuperstein, 1986). In this program, emphasis is on emergent properties of a neural architecture and on neural network dynamics within systems whose effectors may have many degrees

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of freedom and whose mechanical parameters may change due to growth, injury, etc. Our goal is to explain how these systems exhibit such exquisite coordination in many distinct task environments, and how they maintain such coordination during all but the most rapid periods of growth.

Simulations of neural networks for self-organization of spatial maps and continuous autonomous calibration were presented in the book by Grossberg and Kuperstein (1986) on adaptive neural control of ballistic eye movements. The research reported in Bullock and Grossberg (1988a) built on the basis developed in that book, while focusing on automatic trajectory formation and coordination during variable-speed arm movements. In particular, a real-time neural network model, called the Vector Integration to Endpoint, or VITE, Model, was developed and used to quantitatively simulate behavioral and neural data about planned and passive arm movements. In this paper, we extend our application of the VITE model to explain additional neurophysiological and kinematic data on arm movements as well as data about speech articulator movements. In particular, after reviewing a number of basic properties of the model, we discuss data concerning the role of the globus pallidus in movement speed control, the equifinality of movement synergies, rate-dependence of velocity profile asymmetries, and observed variations in the ratio of maximum to average movement velocities. We also note the inability of various other models, notably optimization models, to explain these data, and emphasize the close relationship between a model's computational form and the possibility of correctly identifying and parsing the functional problems that are solved by a complex biological system.

2. EMERGENT INVARIANTS OF THE VITE CIRCUIT

In the VITE model for arm movements, kinematic invariants emerge through network interactions rather than through an explicitly precomputed trajectory. Parallel motor planning occurs in the form of a Target Position Command, or TPC, an array that specifies the lengths to which all arm-controlling muscles intend to move, and an independently controlled GO command, which specifies the movement's overall speed. Automatic processes convert this information into an arm trajectory with invariant properties, notably properties of synchrony among muscle synergists. These automatic processes include computation of a Present Position Command, or PPC, and a Difference Vector, or DV. The DV is the difference between the TPC and the PPC at any time. The PPC is gradually updated

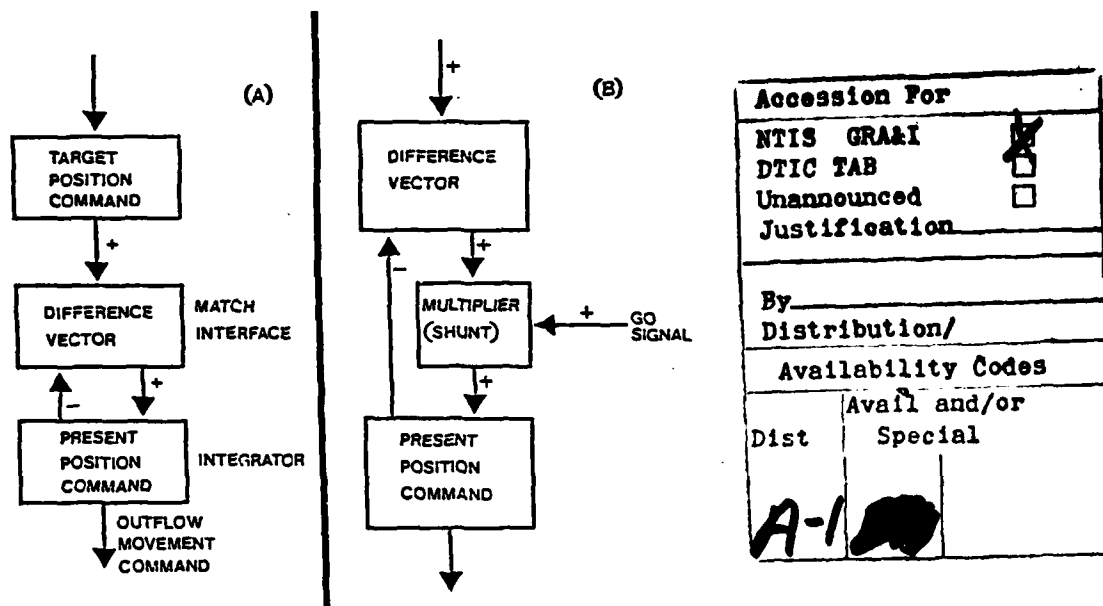


Figure 1. (a) A match interface within the VITE model continuously computes a difference vector (DV) between a target position command (TPC) and a present position command (PPC), and adds the difference vector to the present position command. (b) A GO signal gates execution of a primed movement vector and regulates the rate at which the movement vector updates the present position command.

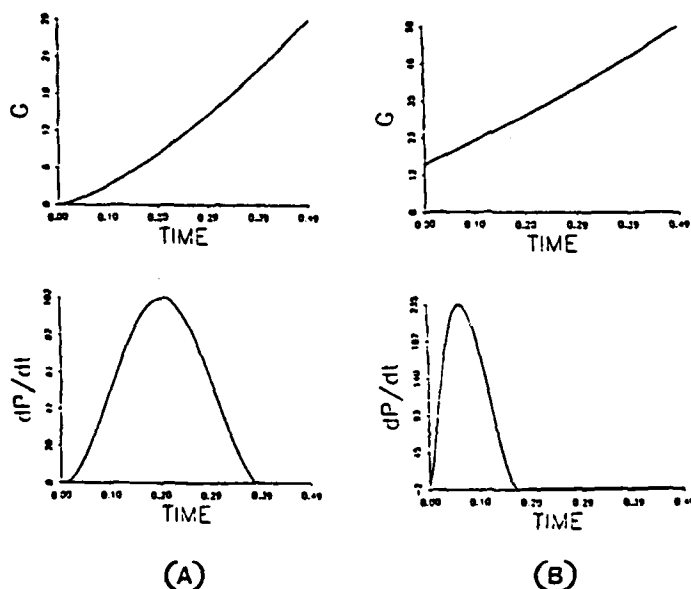


Figure 2. A much higher peak velocity is predicted by the model whenever a target, T , is activated after the GO signal has already had time to grow. (A): The control condition, in which T and the GO signal growth process are activated synchronously. (B): Same T as in (A), but here T was activated after the GO signal $G(t)$ had been growing for 300 msec.

by integrating the DV through time. The time-varying GO signal multiplies the DV before it is integrated by the PPC. The PPC generates an outflow movement command to its target muscle groups (Figure 1). Opponent interactions regulate the PPC's to agonist and antagonist muscle groups at each joint.

In its simplest form, excluding terms expressing opponent interactions, the VITE circuit obeys the equations:

Difference Vector

$$\frac{d}{dt}V_i = \alpha(-V_i + T_i - P_i) \quad (1)$$

and

Present Position Command

$$\frac{d}{dt}P_i = G[V_i]^+, \quad (2)$$

where $[V_i]^+ = \max(V_i, 0)$. Equations (1) and (2) describe interactions of a generic component of a target position command (T_1, T_2, \dots, T_n) , a difference vector (V_1, V_2, \dots, V_n) , a present position command (P_1, P_2, \dots, P_n) , and a time-varying velocity command, or GO signal $G(t)$. The difference vector computes a mismatch between target position and present position, and is used to update present position at a variable rate determined by $G(t)$ until the present position matches the target position. Such a scheme permits multiple muscles, or other motor effectors, to contract synchronously even though the total amount of contraction, scaled by $T_i(0) - P_i(0)$, may be different for each effector (Figure 3).

The VITE circuit is not sufficient in itself to accomplish all the tasks required of a variable-speed variable-load arm movement system. In concert with several parallel circuits, however, it can generate flexible and adaptive trajectories without suffering from the combinatorial explosions and rigid performance of control systems that preplan an entire trajectory. In particular, a key difference between the VITE model and many other model proposals is that its PPC is computed by outflow or feedforward, signals, rather than by inflow, or feedback, signals from the muscles. The process of guaranteeing that the PPC outflow movement command actually moves the arm to a corresponding position are accomplished, in part, by a separate adaptive linearization network that uses outflow-inflow mismatches to generate error signals that adaptively alter the gain of the total outflow movement command (Grossberg and Kuperstein, 1986). The cerebellum has been implicated

TABLE 1
FOR FIXED DURATION (MT), ERROR
GROWS IN PROPORTION TO DISTANCE

MT	DISTANCE	ERROR
.56	10	.084
.56	20	.170
.56	40	.349
.56	80	.700

TABLE 2
FOR FIXED ERROR LEVEL, DURATION (MT)
GROWS LINEARLY WITH DISTANCE DOUBLING

ERROR	DISTANCE	MT
.059	2	.39
.057	4	.49
.058	8	.59
.059	16	.70
.057	32	.80
.059	64	.91

as the locus of adaptive gain change by the manner in which the functional architecture of this model compares with neural data.

The VITE model generates synchronous movements across synergetic muscles by automatically compensating for the different total contractions that each muscle group must undergo. The model accomplishes this in a manner that generates, in quantitative computer simulations, a wide range of properties documented by experimentalists in psychology and neurophysiology (see Georgopoulos, 1986). Among these properties are: Woodworth's law (Table 1 and Woodworth, 1899); the speed-accuracy trade-off function known as Fitts' law (Table 2 and Fitts, 1954); peak acceleration as a function of movement amplitude and duration (Table 3) and isotonic arm movement properties before and after arm-deafferentation in animals deprived of visual feedback (Bizzi, Accornero, Chapple and Hogan, 1984); synchronous and compensatory "central error correction" properties of isometric contractions (Gordon and Ghez, 1987); velocity amplification during target switching (Figure 2 and Georgopoulos, Kalaska, and Massey, 1981); velocity profile invariance across different movement distances (Freund and Büdingen, 1978) and change in profile asymmetry across different movement durations (Figure 3 and Beggs and Howarth, 1972); the automatic compensation for staggered onset times of synergetic muscles (Figure 4 and Hollerbach, Moore, and Atkeson,

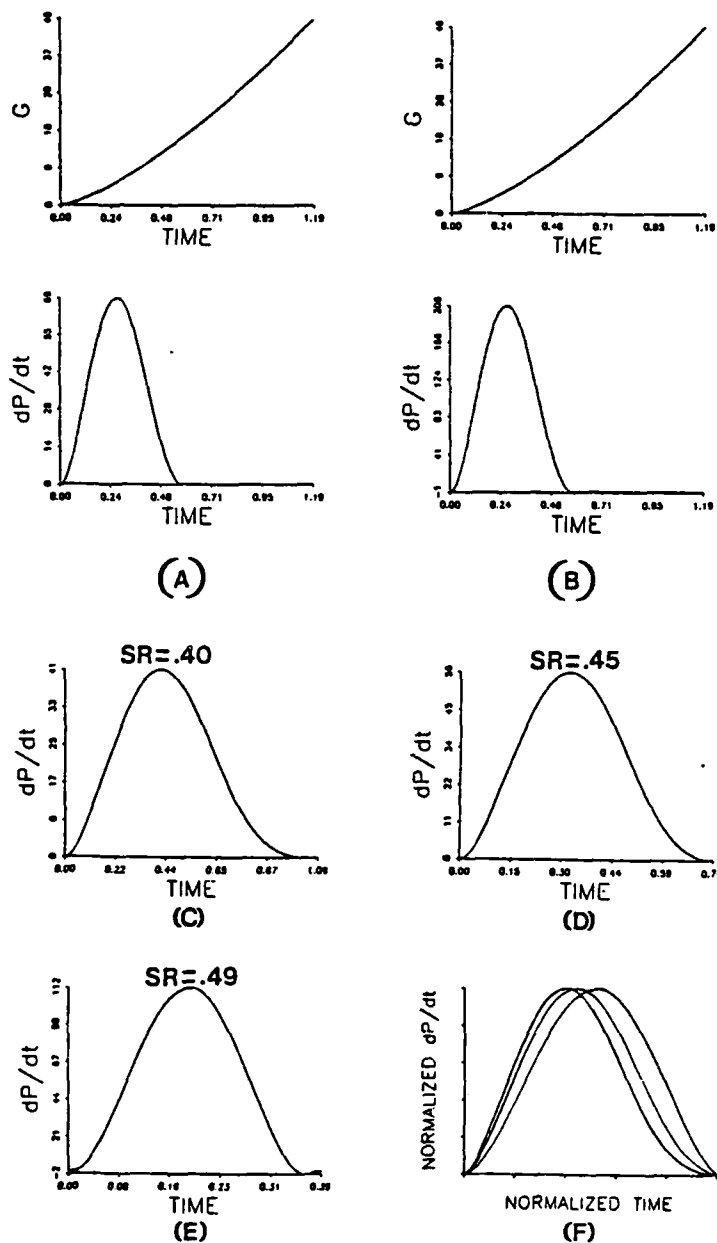


Figure 3. (A,B): With equal GO signals, movements of different size have equal durations and perfectly superimposable velocity profiles after velocity axis rescaling. Shown are GO signals and velocity profiles for 20 and 60 unit movements lasting 500 msec. (C,D,E): Velocity profiles associated with small, medium, and large GO magnitudes result in slow, medium, and fast performance of a 20 unit movement. Each SR value gives the trajectory's *symmetry ratio*; that is, the time taken to move half the distance divided by the total movement duration. These ratios indicate progressive symmetrization at higher speeds, within the range of speeds shown. (F): The velocity profiles shown in (C), (D), and (E) are not perfectly superimposable.

⊥ SYNERGIST BEGINS CONTRACTION
 ↑ SYNERGIST ENDS CONTRACTION

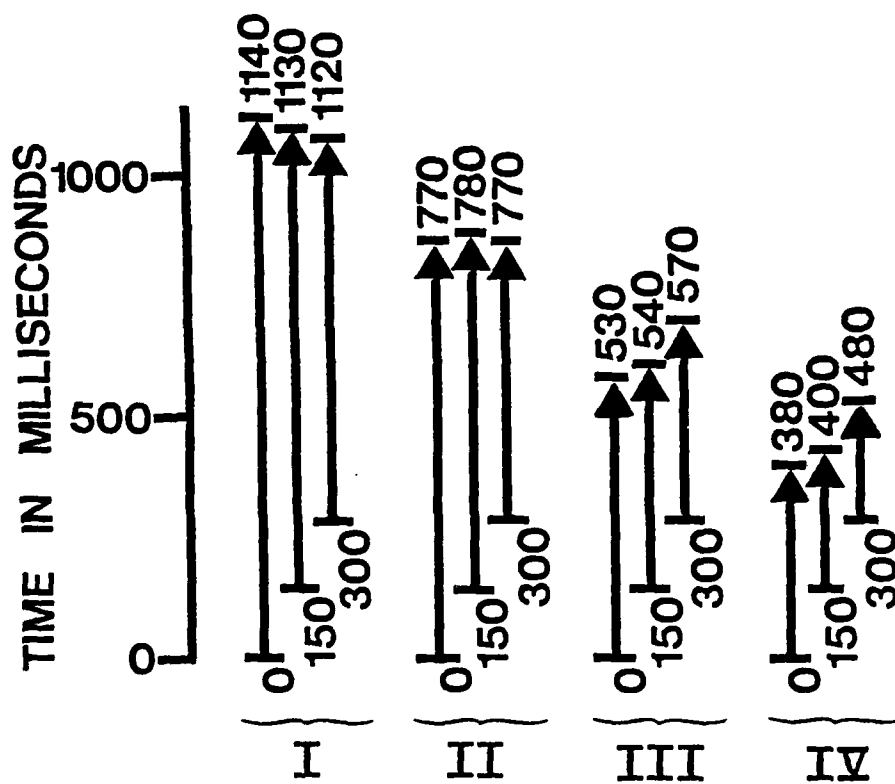


Figure 4. Simulation results showing automatic VITE circuit compensation for contraction-onset-time staggering across components of a synergy. Each block (I, II, III, IV) shows results for a different value (10, 20, 40, and 80, respectively) of the GO signal magnitude.

TABLE 3
A COMPARISON OF FOUR MODELS' ABILITIES
TO PREDICT DATA ON PEAK ACCELERATION (\ddot{P})

DISTANCE	MT	PEAK \ddot{P}	PEAK \ddot{P} SOURCE
20°	.554	397°/sec ²	Bizzi <i>et al.</i> (1984)
60°	.692	1130°/sec ²	(experimental data)
20°	.554	376°/sec ²	Minimum-jerk model
60°	.692	722°/sec ²	(simulation)
20°	.554	399°/sec ²	Minimum-effort model
60°	.692	767°/sec ²	(simulation)
20°	.554	394°/sec ²	VITE model
60°	.692	854°/sec ²	(simulation)
20°	.554	396°/sec ²	VITE ⁺ model
60°	.692	1127°/sec ²	(simulation)

1986); vector cell properties in precentral motor cortex (Georgopoulos, Kalaska, Caminiti, and Massey, 1984; Evarts, 1984); and the inverse relationship between movement duration and peak velocity (Lestienne, 1979).

3. ACTIVELY GATED LEARNING OF TARGET POSITION AND PRESENT POSITION

Beyond their service in trajectory formation, TPC, PPC, and DV computations are needed to actively modulate, or gate, the learning of associative maps between TPC's of different modalities, such as between the eye-head system and the hand-arm system (Figure 5). The gating process prevents learning from occurring except when the PPC is close to the TPC; that is, except when the DV is small. Such gating helps to prevent spurious correlations from being learned, say between a fixed target position of the hand and all present positions which the eye assumes while moving to look at the hand. By using such an intermodality associative map, looking at an object can activate a TPC of the hand-arm system, as Piaget (1963) noted. Then a VITE circuit can translate this latter TPC into a synchronous movement trajectory.

Active gating is also needed to regulate learning of present position commands, notably to prevent such learning from occurring during active arm movements. In particular, an auxiliary circuit, called the Passive Update of Position, or PUP, Model, uses inflow signals to update the PPC during passive arm movements due

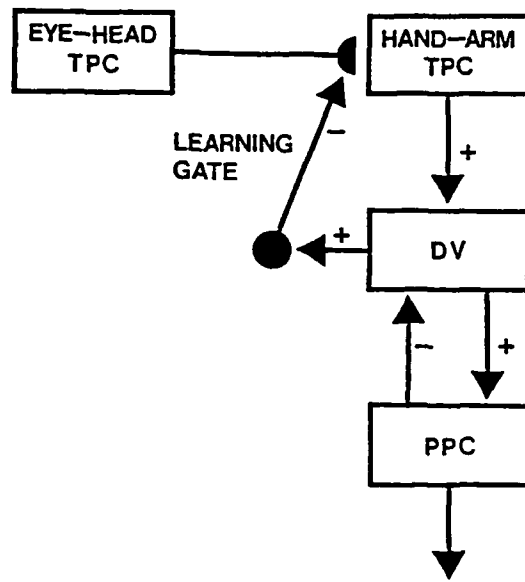


Figure 5. Learning of an intermodal associative transformation between target position maps is gated by a DV process which matches TPC with PPC to prevent incorrect associations from forming between eye-head TPC's and hand-arm TPC's. Learning only occurs when the DV is small.

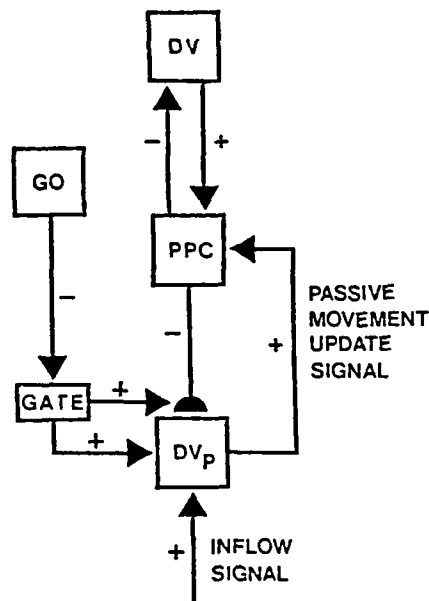


Figure 6. A passive update of position (PUP) circuit. An adaptive pathway $PPC \rightarrow DV_p$ calibrates PPC-outflow signals in the same scale as inflow signals during intervals of posture. During passive movements, output from GO equals zero. Hence the passive difference vector DV_p updates the PPC until it equals the new position caused by any passive movements that may occur due to the application of external forces.

to external forces (Figure 6), but not during active arm movements. Because the scales of outflow position command signals and inflow position sensing signals cannot be assumed to be the same, the PUP circuit incorporates a synaptic modification mechanism for adaptively recalibrating corollary discharges of PPC outflow signals until they are computed in the same numerical scale as the inflow signals to which they are compared. Interactions of outflow and inflow signals are also needed for adaptive linearization of a nonlinear muscle plant (Grossberg and Kuperstein, 1986) and for automatically or predictively adapting to the inertial properties generated by variable loads and velocities (Bullock and Grossberg, 1988b). The equations of a typical PUP circuit are:

Present Position Command

$$\frac{d}{dt}P_i = G[V_i]^+ + G_p[M_i]^+ \quad (3)$$

Outflow-Inflow Match

$$\frac{d}{dt}M_i = -\beta M_i + \gamma I_i - z_i P_i \quad (4)$$

Adaptive Gain Control

$$\frac{d}{dt}z_i = \delta G_p(-\epsilon z_i + [M_i]^+). \quad (5)$$

Equation (3) supplements equation (2) with an update signal $G_p[M_i]^+$ that is turned on only when the passive gating function, or "pauser" signal, G_p becomes positive in the passive, or postural, state. Function z_i in (5) is an long term memory trace, or associative weight, which adaptively recalibrates the gain of outflow signals P_i until they are in the same scale as inflow signals γI_i in (4).

In summary, offset of the GO signal within the VITE circuit enables a pauser signal within the PUP circuit to drive its learning and reset functions. Such pauser-modulated learning during mismatches has been suggested to occur in several adaptive sensory-motor control circuits (Grossberg and Kuperstein, 1986).

4. PHYSIOLOGICAL EVIDENCE FOR GO-SIGNAL PATHWAY IN GLOBUS PALLIDUS

Because the VITE model proposes that trajectories are generated as the arm tracks the evolving state of a neural circuit, the model can be tested in two ways:

by comparing trajectories of the neural circuit's output stage with actual arm trajectories, and by checking for the existence of the neural components postulated in the model.

In Bullock and Grossberg (1988a) we reviewed evidence of Georgopoulos and his colleagues that cell populations in precentral motor cortex could be analysed as an *in vivo* analogue of model DV stage neurons. Additional physiological support for the VITE model comes from recent experiments involving lesions and electrical stimulation of the basal ganglia. Data from a set of experiments by Horak and Anderson (1984a, 1984b) are consistent with the interpretation that the internal segment of the globus pallidus is an *in vivo* analogue of the VITE model's GO-signal pathway.

An *in vivo* candidate for a GO-signal pathway must pass three tests. First, stimulation at some site in the proposed pathway must have an effect on the *rate* of muscle contractions. Second, it must have this effect without affecting the *amplitude* of the contractions. Thus stimulation should have no effect on movement accuracy. Third, this rate-modulating effect should be non-specific: it should affect all muscles that are typically synergists for the movement in question.

The studies conducted by Horak and Anderson (1984a, 1984b) addressed these issues. Horak and Anderson (1984a) showed that "when neurons in the globus pallidus were destroyed by injections of kainic acid (KA) during task execution, contralateral arm movement times (MT) were increased significantly, with little or no change in reaction times (p.290)." This satisfies the rate criterion. Moreover, the rate of motor recruitment was depressed "in all the contralateral muscles studied at the wrist, elbow, shoulder, and back, but there were no changes in the sequential activation of the muscles (p.20)." This satisfies the non-specificity criterion. Finally, the authors also noted that "animals displayed no obvious difficulty in aiming accurately ... they did not miss the 1.5-cm target more often following KA injections, and there was no noticeable dysmetria around the target (p.300)." This satisfies the accuracy criterion.

Horak and Anderson (1984b) used an electrical stimulation paradigm instead of a lesion paradigm. They found that "stimulation in the ventrolateral internal segment of the globus pallidus (GP_i) or in the ansa lenticularis reduced movement time, whereas stimulation at many sites in the external pallidal segment (GP_e), dorsal (GP_d), and putamen increased movement times for the contralateral arm

(p.305).” Once again, these effects were non-specific: “no somatotopic effects of stimulation were evident. If stimulation at a site produced slowing, it produced a depression of activity in all the muscles studied. Even stimulus currents as low as 25 μA affected proximal as well as distal muscles, flexor as well as extensor muscles, and early- as well as late-occurring activity (p.309).”

In the VITE model, activation of the GO-signal pathway produces movement only if instatement of a TPC different from the current PPC leads to the computation of a non-zero DV. In agreement with this property, Horak and Anderson (1984b) observed that “stimulation at sites that speeded movements did not induce involuntary muscle activation in resting animals nor did it change background EMG activity prior to self-generated activity during task performance (p.313).” In Bullock and Grossberg (1988a) we noted that “very rapid freezing can be achieved by completely inhibiting the GO signal at any point in the trajectory”. This property of the model has also been shown to be a property of the GP system. In particular, Horak and Anderson reported that “stimulation with 50 or 100 μA at ... sites ventral and medial to typical GP; neuronal activity completely and immediately halted the monkey’s performance in the task (p.315).” Taken together, their experiments led Horak and Anderson (1984b) to conclude that “the basal ganglia ... determine the speed of the movement” (p.321).

5. ENSURING EQUIFINALITY IN SPACE AND TIME

The striking correspondence between the experimental results of Georgopoulos *et al.* and of Horak and Anderson and the theoretical predictions of the VITE model regarding separate DV and GO-signal processes is important because it supports the hypothesis that motor systems, like sensory systems, implement a factorization of pattern and energy (Grossberg, 1970, 1978a, 1982). In the motor system, this factorization means that a movement’s speed (“energy”) can be scaled up or down over a wide range without disrupting the movement’s direction or spatial endpoint (“pattern”). Moreover, by using a GO-signal that grows gradually during the movement time (as exemplified in Figure 2), all synergists will complete their contractions at approximately the same time even if movement onset times of different synergists are staggered by a large amount (Figure 4). These properties of the model, together with the strong evidence for separate DV and GO-signal pathways *in vivo*, provide a basis for understanding how primates can achieve space-time equifinality—all synergists reaching their length targets

at equal times—yet retain separate control of rate and position. Note that rate-control models relying on *static* stiffness adjustments (e.g., Cooke, 1980) lack the critical temporal-equi-finality property.

Several other important automatic compensatory properties emerge from interactions between the DV and GO processes. In Bullock and Grossberg (1988a), we noted that in addition to compensating for muscles that begin to contract at staggered onset times, the VITE circuit automatically compensates for changes of target position during the movement time. In particular, Figure 2 shows that the model generates the amplification of peak velocity that occurs during target-switching experiments (Georgopoulos, *et al.*, 1981).

Such an amplification of velocity facilitates reaching the target after an incorrect initial TPC is replaced by an updated TPC. This speed-up occurs “on-the-fly.” It is not preprogrammed, but is rather an automatic emergent property of VITE circuit interactions.

The VITE circuit can accomplish such compensations because changes in the TPC during target switching, or differences in onset times of contraction across muscles in a synergy, or updating of a PPC via inflow signals during an externally caused movement—all these circuit perturbations *flow through* the system via dynamic real-time computations. For example, during a target-switching experiment, first there is quick reset of the TPC, and then more gradual compensatory change of the DV and the DV-GO signal product that controls movement rate.

Similar catch-up phenomena are known to exist in the domain of speech motor control. Abbs, Gracco, and Cole (1984) have argued that such speech compensations cannot be explained by more common compensatory mechanisms such as stretch reflexes. Though a full treatment of how the VITE circuit contributes to an explanation of these data will not be given here, other data suggestive of a VITE circuit role in speech are treated in Sections 6 and 7.

6. RATE-DEPENDENT ASYMMETRY IN VELOCITY PROFILES

A distinctive prediction of the VITE model is that velocity profiles will generally be asymmetrical about the movement-time midpoint, and that their degree and direction of asymmetry will vary as a function of movement time (MT). Such a dependence of asymmetry on MT has been known empirically since the work of Beggs and Howarth (1972), whose results were modelled via the series of sim-

ulations shown in Figure 3C, D, and E. Recently, Zelaznik, Schmidt, and Gielen (1986) have shown that within a faster range of speeds ($MT < 250$ msec.), the direction of asymmetry can reverse from that observed in the medium-to-slow speed range studied by Beggs and Howarth. Table 4 shows results from a series of VITE model simulations that replicate the results reported in Zelaznik *et al.* (1986). The T-SR values (time symmetry ratios) were calculated as in Figure 3: the time required to move half the total distance was divided by the time required to move the entire distance. The D-SR values (distance symmetry ratios) were calculated as in Zelaznik *et al.* (1986); namely, the distance traversed at the time of the arm's peak velocity was divided by the total movement distance.

Model parameter settings were the same as those used in Bullock and Grossberg (1988a). To match the Zelaznik *et al.* measurement conditions as closely as possible, measurements of duration and distance in the simulation runs began when velocity first exceeded a 4 cm/sec threshold and ended when velocity fell below a 4 cm/sec threshold (see the ϵ_2 columns). As can be seen in Table 4, model-generated D-SR values for movement times between 250 and 125 msec. exhibit the direction of asymmetry (all D-SRs $> .5$) observed by Zelaznik *et al.* Moreover, their range (.54 to .58) closely matches the observed range (.54 to .60). Finally, the ordering of the model-generated values is also consistent with the overall pattern established by the observed values: "the distance traversed at the time of maximum velocity was inversely related to MT" (Zelaznik *et al.*, 1986, p.361).

Several additional remarks should be made about the T-SR and D-SR values shown in Table 4. First, these values are invariant under changes of TPC and initial DV. That is, they vary with movement rate, but not movement amplitude. Second, both these statistics are stable despite small shifts in the velocity threshold used to begin and end measurements of a given movement (compare the ϵ_1 and ϵ_2 columns). Third, it can be seen from the table that the T-SR statistic is more sensitive to change in VITE velocity profiles than the D-SR statistic, which even shows a slight violation of monotonicity. Because Zelaznik *et al.* also appeared to have difficulty proving replicability of the small D-SR differences they initially observed (compare their Experiment 1 and Experiment 2 results), we suggest that future experiments concerned with rate-dependent asymmetry use the T-SR statistic.

Two final points concern the generality and origin of rate-dependent asym-

TABLE 4

KINEMATIC VARIABLES AS A FUNCTION OF THE GO-SIGNAL SCALAR (G_o) AND THE VELOCITY THRESHOLD (ϵ_i)

G_o	V_{max}	Distance		Duration		V_{max}/V_{avg}		T-SR		D-SR	
		ϵ_1	ϵ_2	ϵ_1	ϵ_2	ϵ_1	ϵ_2	ϵ_1	ϵ_2	ϵ_1	ϵ_2
10	38.2	19.9	19.6	.96	.84	1.84	1.63	.44	.45	.50	.50
20	52.4	20.0	19.8	.71	.63	1.86	1.67	.45	.46	.49	.49
40	72.8	20.2	20.1	.50	.47	1.80	1.70	.46	.47	.51	.51
80	102.5	20.6	20.6	.36	.34	1.79	1.69	.50	.50	.53	.53
160	145.7	21.5	21.5	.27	.26	1.79	1.73	.51	.51	.54	.54
320	207.7	22.9	22.9	.20	.20	1.81	1.77	.55	.54	.57	.57
640	295.8	24.9	24.8	.16	.15	1.84	1.73	.55	.55	.58	.58
1280	417.9	27.2	27.2	.12	.12	1.85	1.77	.56	.57	.58	.58

Note: Mean V_{max}/V_{avg} values are 1.82 and 1.71 in columns ϵ_1 and ϵ_2 , respectively. $\epsilon_1 = 1$ unit/sec. $\epsilon_2 = 4$ units/sec. TPC = 20 for all simulations.

metries. Although the VITE model was developed for arm movements, there are many reasons to believe that it is applicable to speech motor control. Like reaching movements, speech movements are subject to continuous rate variations, and higher speaking rates lead to poorer accuracy. Recently it has been shown that speech movements also exhibit unimodal velocity profiles that are comparable to those shown in Figure 3 for arm movements and Ostry, Cooke, and Munhall (1987) have reported strong "evidence for ... a rate dependent asymmetry in the velocity of orofacial movement, under a variety of conditions" (p.44). Moreover, because the asymmetry was true of both speech and non-speech movements (i.e. was not task-specific) the authors concluded that "the most promising alternatives to account for the asymmetry of form seem to be articulator biomechanics and/or neural control" (p.45) rather than speech-specific task demands. Because the rate-dependent asymmetry is common to several biomechanically dissimilar systems, we are inclined to place more weight on an explanation in terms of a neural controller, viz., the VITE circuit. The asymmetric velocity profiles of the VITE model coexist with the model's asymmetric DV profiles, as equation (2) shows. The asymmetric DV profiles are compared in Bullock and Grossberg (1988a) with neurophysiological evidence from the Georgopoulos laboratory demonstrating asymmetric vector

cell profiles in precentral motor cortex.

7. A COMMON KINEMATIC INVARIANT IN SPEECH AND ARM MOVEMENTS: V_{max}/V_{avg}

One kinematic measure that is now frequently used in the experimental literature on arm and speech articulator movements (Hasan, 1986; Munhall, Ostry and Parush, 1985; Ostry and Cooke, 1987; Ostry *et al.*, 1987; Soechting, 1984) is the ratio of a movement's maximum velocity (V_{max}) to its average velocity (V_{avg}). This ratio has been found to be virtually constant across a wide range of movement speeds and distances in both the speech and reaching domains. Moreover, the absolute values of the ratios found for speech and reaching movements—about 1.7 and 1.9, respectively—are close enough to invite speculation that this kinematic invariant might be a “signature” of a generative mechanism common to the two domains.

The VITE model predicts a mean value of 1.95 (range 2.18 to 1.86, with a .001 unit/sec velocity threshold; predicted ranges and means for two other thresholds are shown in Table 4), which stands exactly at the midpoint of the empirical range reported by Hasan (1986) for arm movements. Other models of arm trajectory formation predict similar values: Hogan's (1984) minimum jerk model predicts 1.88, whereas Hasan's minimum effort model predicts 1.97. Why are both empirical and predicted values for discrete arm movements (1.9) larger than commonly observed values for speech movements (1.7)?

A simple hypothesis is that the discrepancy is, at least partly, due to a truncation effect. Note that if you truncate the tails of a bell-shaped velocity profile, and if you measure distance and duration relative to the truncated profile, then V_{max} remains the same while V_{avg} increases, relative to values calculated from a full (un-truncated) profile. Thus the ratio V_{max}/V_{avg} varies inversely with the degree to which the velocity profile's edges get clipped. This is illustrated in Table 4 where the ϵ_2 column represents a more severe truncation than the ϵ_1 column.

We consider two kinds of evidence that truncation does contribute to the arm vs. speech discrepancy. First, Soechting (1984) reported a speech-like value of V_{max}/V_{avg} (1.75) in an arm experiment. However his experimental procedures can explain this anomalous value as an example of a truncation artifact. As he noted, “Movement time T was defined as the interval between the time at which wrist velocity began to exceed 10% of its maximal value and the time of contact with

the target." This meant that movements had already achieved velocities of from 6.5 to 16.5 cm/sec before measurement began. By omitting these small velocities from the average, the average velocity was spuriously inflated. (In addition to causing an underestimation of V_{max}/V_{avg} , this severe truncation led Soechting to mistakenly conclude that velocity profiles from fast and slow movements were superimposable after time and velocity normalization—contrary to the findings of Beggs and Howarth [1972], Moore and Marteniuk [1986], and Zelaznik *et al.* [1986] regarding rate-dependent asymmetry.)

Second, Munhall *et al.* (1985) have shown that the ratio varies across speech conditions. By examining their tables of mean values as a function of speech condition, it is possible to test the truncation hypothesis. However, in this case we are speaking not of a truncation artifact introduced by experimental procedures, but of a "real" truncation caused by switching to a new TPC before the prior DV has been completely zeroed. We believe that such non-artifactual truncations are the rule in medium- and fast-paced continuous speech.

Were such normative truncation the source of the lower V_{max}/V_{avg} values, then speech V_{max}/V_{avg} values should be higher (and closer to discrete arm movement values) when the subject speaks more slowly, when the subject places greater stress on a syllable, and when the consonantal target position must be briefly held (as in a fricative). Consistent with this expectation, inspection of the Munhall *et al.* (1985, Table 2, p.464) data reveals the highest mean V_{max}/V_{avg} value, 1.83, for the slow, stressed, fricative condition, and the lowest mean value, 1.66, for the fast, unstressed, stop consonant condition (compare Table 4 simulation means). A more recent experiment (Ostry *et al.*, 1987), though conducted for other reasons, provided a more direct test of the truncation hypothesis by examining the V_{max}/V_{avg} ratio for both continuous and discrete elbow, tongue, and jaw movements. The data show that for both tongue and jaw lowering, and for elbow movements, "the ratio was greater in the discrete than in the continuous condition ($p < .001$)" (p.41).

In summary, the small but reliable discrepancy between V_{max}/V_{avg} values observed in studies of discrete reaching vs. continuous speech can be plausibly explained as a truncation effect. Thus this discrepancy should not block further pursuit of the hypothesis that a common mechanism explains trajectory formation in both systems (see also Cohen, Grossberg, and Stork, 1988).

8. KINEMATIC PROPERTIES AND COMPETING MODELS OF TRAJECTORY FORMATION

The abundance of information on kinematic properties of planned point-to-point arm movements has prompted the formulation of a number of alternative models of trajectory formation. Many of these models—such as the minimum jerk model (Hogan, 1984) or the minimum effort model (Hasan, 1986)—have attempted to rationalize kinematic properties as consequences of optimizations. Thus Hogan proposed that the bell-like shape of observed velocity profiles indicates an optimization for smooth motions, whereas Hasan proposed that the same shapes reflect an optimization that minimizes the “psychological effort” required to produce movement.

Two types of problems—empirical and conceptual—attend such optimization models. The empirical problem is exemplified by the relatively poor predictions made by these models in Table 3, just one illustration that such models often fail quickly when asked to accommodate kinematic properties, such as acceleratory extrema, which are not tightly linked to the optimized variables. Other failures include their inability to explain such phenomena as the rate-dependent asymmetry of velocity profiles (Section 6) and the anomalous amplification of peak velocity during target switching (Section 5).

Yet another difficulty derives from the observed variability in V_{max}/V_{avg} . As Ostry *et al.* (1987) noted, the V_{max}/V_{avg} values found for continuous movement were close to those predicted by a minimum-energy model (Nelson, 1983) whereas the values found for discrete movement were close to those predicted by a minimum-jerk model (Hogan, 1984). Rather than attempting to rationalize these outcomes, which are contradictory within any single optimization framework, we interpret such multiple failures as strong evidence that optimization models do not incorporate the rate-limiting designs that generate the parametric structure of arm movement data.

The conceptual problem (see also Grossberg, 1978b, 1982; Jacob, 1977; Partridge, 1982) with most optimization models is their lack of constituents that bear any resemblance to the constituents of real biological systems. We believe it is this conceptual problem that leads to the empirical problem: If the constituents are not correct, then the many emergent properties of the ensemble other than the one explicitly designed in (the one optimized for) will also typically be wrong.

In contrast to these optimization models, the VITE model of trajectory formation assumes that the modern neural system for limb movements includes the minimal anatomy that affords coordinated use of an array of muscles to smoothly cancel the distance between a current and desired motor state, and to maintain synchrony of activity across the array despite large differences in overall movement rate and even larger changes in the contraction rates of individual synergists. To emphasize this basic design is not to deny that evolutionary selection pressures implicitly identified in various optimization models may also have been operative. For example, selection favoring smoother movements may have tuned parameters such as the size and rate of change of the GO-signal during the movement interval.

9. CONCLUSION

The VITE model represents a simple but robust solution to the problem of generating flexible and synchronous goal-oriented movement commands capable of compensating for variations in initial position, desired target position, desired movement speed, and onset-asynchrony. Computer simulation studies of the circuit's emergent properties demonstrate that it provides a compact quantitative explanation of a large behavioral database. Brain analogues of two key circuit constituents have been identified, each on the basis of multiple functional criteria.

The VITE model is but one of several circuits (witness the PUP circuit of Section 3) that are being developed to explain how humans and related mammals achieve dexterous control of goal-oriented limbs. These circuits provide a quantitative instantiation of the functional problems and design principles that have been identified through analysis of the large interdisciplinary data base concerning eye, arm, and speech articulator movements. The manner in which these different circuits globally parse this problem space is no less important than the properties of the individual circuits themselves, since if one tries to solve the wrong set of problems one can never understand in a principled way the dynamics of a complex biological system.

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